No skin off YOUR back!

Amphibians invest in parenthood in a variety of ways including hiding, guarding, transporting, and feeding their progeny. While the provision of nutrient-dense yolk is the primary food for many oviparous caecilian species, young of the Kenyan caecilian *Boulengerula taitanus* were observed by Kupfer and his colleagues to peel and eat the outer layer of their mother's skin. Examination of the stomach contents of young showed the ingested skin was sufficient for growth of up to 11% of their length in one week. The outer layer of epidermis of brooding females, which appears paler than that of non-brooding females, contains cells that are more voluminous and full of vesicles than the typical squamous keratinized cells. This epidermis results from elongation of the epithelial cells as opposed to increases in cell number. Interestingly, this report is the first documentation of the use of fetal-like teeth in oviparous caecilians. See the online video. (*Nature* 440:926–9, 2006)

Itch induces T-cell tolerance

T-cell anergy has been proposed as a mechanism of peripheral T-cell tolerance, a process necessary to stunt Th2 responses that, if unchecked, cause asthmatic and allergic symptoms. Because E3 ubiquitin ligases are upregulated in anergic T cells, Venuprasad and colleagues used a mouse model and stimulated T cells *in vitro* to demonstrate that one such ligase, Itch, is specifically important for Th2-cell anergy. Tolerized Th2 cells displayed increased levels of Itch, and levels of JunB, a transcription factor involved in Th2 differentiation and cytokine production, were reduced. JunB was not reduced in Itch-deficient cells, which indicates that Itch is involved in ubiquitination-mediated JunB degradation. Like Itch-deficient cells, cells lacking mitogen and extracellular kinase kinase 1 (MEKK1) and c-Jun N-terminal kinase 1 (JNK1), which activate Itch via TCR signaling, were resistant to induction of tolerance. Thus, MEKK1 and JNK1 initiate Itch-mediated degradation of JunB to induce Th2 tolerance. Furthermore, Itch deficiency results in airway inflammation, confirming the biological significance of this pathway. (*J Clin Invest* 116:1117–26, 2006)

O-glycosylation boosts cell adhesion

Plakoglobin, a member of the armadillo family of cell adhesion and signaling proteins, is a component of both adherens junctions and desmosomes. This protein is modified post-translationally by phosphorylation and *O*-glycosylation, although the consequences of the latter remain unclear. *O*-glycosylation also modifies transcription factors, nuclear pore proteins, cytoskeleton components, and enzymes. The enzyme responsible for this modification, the UDP-*N*-acetylglucosamine polypeptide β-*N*-acetylglucosamine transferase (OGT), was recently cloned and expressed in murine keratinocytes by Rubenstein's group in order to examine the role of this modification in cell–cell adhesion. Plakoglobin levels and stability were increased in these OGT-overexpressing cells. In addition, the cells exhibited greater cell–cell adhesion in a culture-based assay. Immunofluorescence microscopy demonstrated greater membrane localization by adherens junction and desmosome proteins after overexpression of OGT. Together, these results demonstrate that *O*-glycosylation functions post-translationally to increase the stability of plakoglobin and, in turn, increases the cell–cell adhesion of keratinocytes. (*J Biol Chem* 281:12786–91, 2006)

Education as medicine for eczema

The prevalence of eczema (atopic dermatitis), a chronic skin disease that begins in infancy or early childhood, is increasing in industrialized nations. The clinical symptoms of this relapsing disease burden the entire family of the affected child. Staab and colleagues demonstrated that weekly group educational sessions provided long-lasting benefits to adolescent patients and parents of children with the disease. Approximately 992 families with children ranging in age from 3 months to 18 years participated in this multicenter randomized controlled study. Families that participated in the educational programs reported decreased severity of symptoms and improved quality of life 1 year after commencement of the study. These results suggest that a standardized multidisciplinary educational program could be an important addition to the symptom-oriented therapeutic approach for managing eczema. (*BMJ* 332:933–8, 2006)

Epidermis renewal depends on slug

During terminal differentiation, keratinocytes in the epidermal basal layer halt their cell cycle progression and migrate and form the differentiated suprabasal layers. Integrins play an important role in migration, proliferation, and differentiation. In fact, disruption of the interactions between integrins and the extracellular matrix initiates terminal differentiation. The Snail family of transcriptional repressors regulates genes involved in cell adhesion and migration. In addition, Snail family members, including Slug, are upregulated in multiple human tumors. Recently, Turner and colleagues used conditional activation of Slug in keratinocytes to demonstrate that Slug represses E-cadherin and β1, β4, and α3 integrin via binding to the promoter E-box. Activation of Slug dramatically decreased keratinocyte attachment. Furthermore, Slug activation limited cell proliferation in a manner consistent with cell cycle arrest but, interestingly, did not induce cell differentiation. Thus, repression of E-cadherin and integrin proteins has significant biological repercussions for keratinocyte proliferation, a critical process in the self-renewal properties of the epidermis. (*J Biol Chem* doi:10.1074/jbc.M509731200, May 17 2006)

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